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ARCHIVES OF PEDIATRICS

A MONTHLY DEVOTED TO THE

DISEASES OF INFANTS AND CHILDREN

JOHN FITCH LANDON, M.D., Editor

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With Special Emphasis on Factors Involved in the Realistic Management of the Child with Cerebral Palsy.
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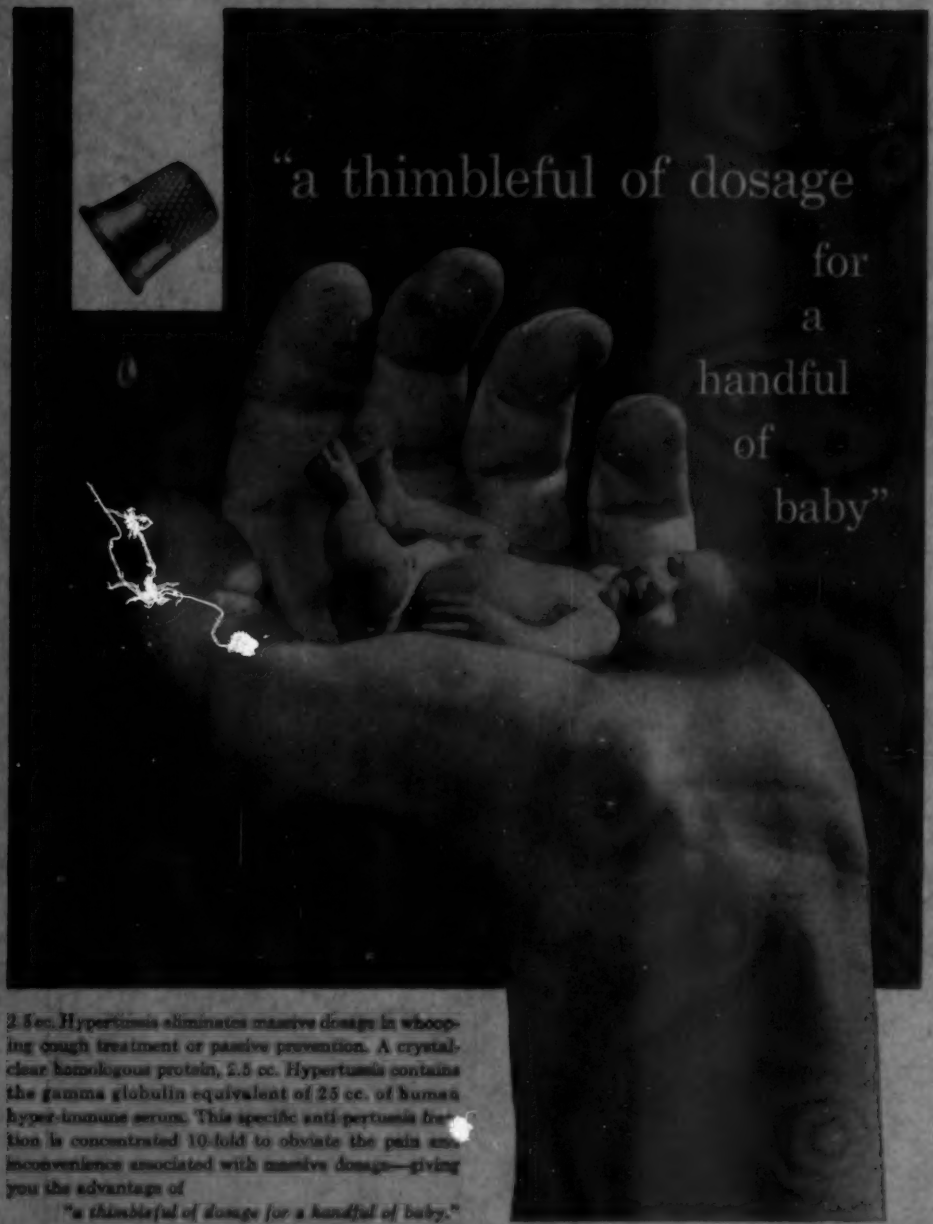

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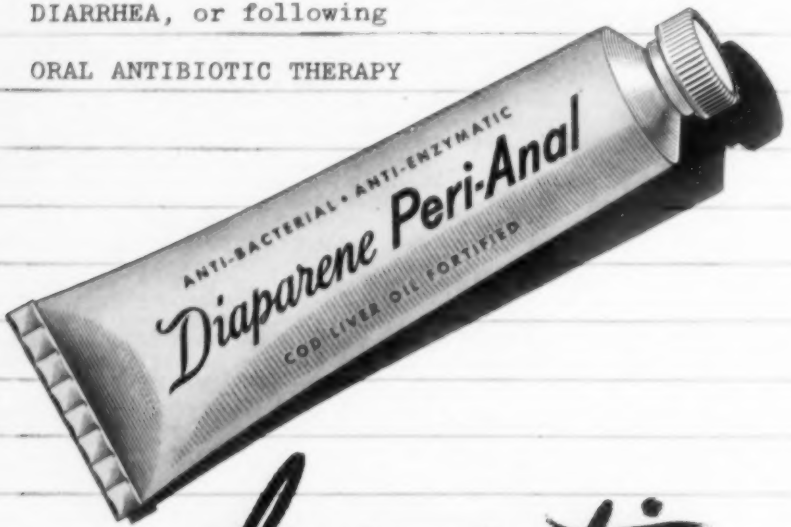

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1. Wood, H. F., and McCarty, M.: Am. J. Med. *17*:768 (Dec.) 1954. 2. Adams, F. H.: J.A.M.A. *156*:1319 (Dec. 4) 1954. 3. Stollerman, G. H., et al.: Am. J. Med. *15*:645 (Nov.) 1953. 4. McEwen, C.: M. Clin. North America (Mar.) 1955, to be published. 5. Elster, S. K.; Wood, H. F., and Seely, R. D.: Am. J. Med. *17*:826 (Dec.) 1954.

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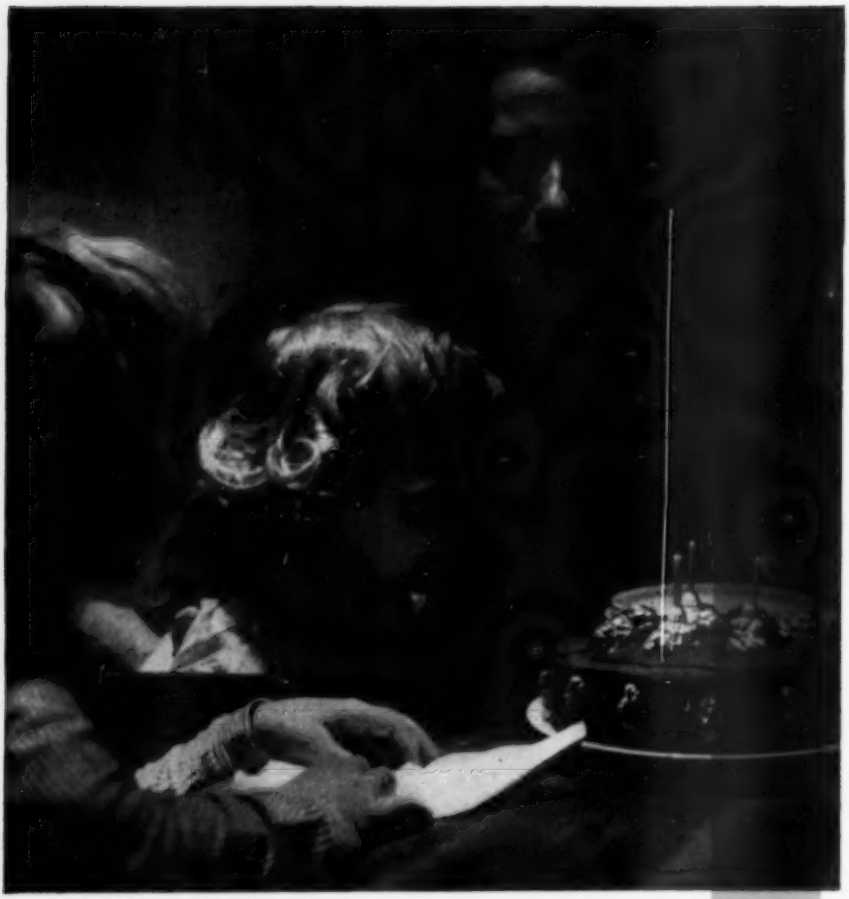

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PEDIATRIC REHABILITATION

WITH SPECIAL EMPHASIS ON FACTORS INVOLVED IN THE REALISTIC MANAGEMENT OF THE CHILD WITH CEREBRAL PALSY*

LOUIS A. SUSCA, Ph.D., M.D.
New York.

I. INTRODUCTION

INCLUDING THE ETIOLOGY AND INCIDENCE OF CEREBRAL PALSY AND THE MANY PROBLEMS PRESENTED BY THIS NEUROLOGIC CONDITION

Cerebral palsy ("spastic diplegia," and "Little's disease") may be defined simply as "paralysis due to a lesion of the brain." Spastic diplegia, more specifically, refers to a spastic paralysis of the legs, due to organic changes in the infantile brain, such as diffuse degeneration or atrophic lobar sclerosis, malformations, and developmental defects, and microscopic cellular alterations. Spastic diplegia is sometimes associated with convulsions and mental deficiency.

Various portions of the infantile brain may be so affected as to produce spasticity (cerebral cortex involvement), athetoid movements (basal ganglia involvement), ataxia and incoordination (cerebellar damage), or rigidity and tremors (diffuse lesions). As early as 1853, Little published a paper entitled: "On the Nature and Treatment of Deformities of the Human Frame," in which the causes for such brain lesions were stated to be of an obstetric nature. Little believed that abnormal parturition, difficult labors, and asphyxia were contributing factors towards the development

*From the New York Medical College, Flower and Fifth Avenue Hospitals, New York.

of cerebral palsy. Osler, in 1889, studied 17 cases of cerebral palsy, and concluded that intracranial hemorrhage at birth was the cause. Since 1938, emphasis has been placed on birth injuries, including asphyxia, as the cause for the paralysis observed in children with cerebral palsy.

TABLE 1.

Historical Aspects as to Obstetrical Causes of Cerebral Palsy

Author	Year	Possible Cause
1. Little	1853-1862	Influence of abnormal parturition, difficult labors, premature birth, asphyxia neonatorum.
2. McNutt	1885	Meningeal hemorrhage.
3. Freud, Sigmund	1897	Intrauterine disease.
4. Osler, Sir Wm.....	1889	Intracranial hemorrhage.
5. Guthrie	1913	Association of kernicterus with choreiform movements.
6. Collier	1924	Pulmonary degeneration of neurones to unknown causes.
7. Yannet and Zimmerman.	1935	Relation of icterus and cerebral sequellae.
8. Alpers and Marcovitz ..	1938	Birth injury may produce palsy.
9. Schreiber	1938	Cerebral lesions considered congenital may be due to asphyxia.
10. Penfield and Erickson..	1941	Certain lesions of birth and early infancy capable of producing microgyria.
11. Friedman and Courville.	1941	Atrophic lobar sclerosis and partial cerebral agenesis.
12. Benda	1945	Supports birth injury idea; symmetrical paralysis may have asymmetrical lesion.

Faber, in 1947, stated that factors causing cerebral damage in children could be divided into three groups, as follows:

(1) Prenatal—especially abruptio placentae and placenta previa. (2) Paranatal—especially anesthetics and sedation and induced labor. (3) Postnatal—such as measles, meningitis and pertussis.

It is apparent, therefore, that a large number of factors may play a role in the etiology of cerebral palsy. A study of the historical aspects as to obstetrical causes of cerebral palsy emphasizes this viewpoint easily (Table 1).

The exact incidence of cerebral palsy in the United States was set forth by Levin, Brightman and Burth in 1952. They stated the incidence as 5.9 per 1,000 births, or 6 per 100,000 population.

These figures could be made more accurate if physicians in all hospitals would establish clinics for follow-up of infants who are apneic or asphyxiated at birth, as these authors believed also that there was a definite correlation between cerebral palsy and these factors. It is interesting, also, to note that such an incidence, i.e., 5.9 per 1,000 births, is equal almost to the combined deaths from birth injury and anoxia at the Chicago Lying-In Hospital, for 1952.

Medical research has revealed that children with cerebral palsy present unique problems in development, learning and adjustment. It is difficult to minimize to the greatest extent possible, each handicap of the child with cerebral palsy, and at the same time to give that child the medical care, education and training consistent with each child's specific needs. This problem is further amplified in scope because of its relative newness in the field of pediatrics.

Total care in the field of rehabilitation involves a team rather than one person. Physicians, psychologists, physical therapists, occupational therapists, teachers, training supervisors and counselors—all are involved. However, rehabilitation requires not only that each of these individuals participates actively, but also, one other—this person is the patient himself. This is true regardless of the age of the patient (other than those considered during infancy). Furthermore, the physician, especially the pediatrician, must be prepared and willing to meet the patient more than halfway, with all the optimism, emotional objectivity and maturity at the pediatrician's command.

Additional important factors, which the pediatrician should check in his attempt to manage such children, involve knowledge as to the preparation of the remaining members of the rehabilitation team. Teachers working with such patients should understand the psychopathology of the brain-injured child. The teacher's training should include orientation in child development, as well as experience in teaching. The pediatrician should interest himself in the goals of the teacher of his patient, and the pediatrician should help the patient develop interests and understandings pertaining to the world in which the patient lives.

To be complete, the team should include the family of the cerebral palsied child. The greatest asset to the training of such a child is his parents. The pediatrician should help the family obtain

adequate knowledge and emotional stability regarding problems associated with the cerebral palsied child in the home. The pediatrician of the future will learn, as a result of this need, the merits of group counseling.

It is important that facilities for diagnosis be made available to each child with cerebral palsy. If this cannot be done on an individual basis, then the pediatrician should be able to refer his patient to an adequate diagnostic center. Such a center will include neurologist, psychiatrist, opthalmologist, otologist and otolaryngologist, as well as pediatricians. Furthermore, the pediatrician should keep in mind the fact that a psychological examination, involving only the I.Q., does *not* portray the mental status of the cerebral palsied child who also has unusual sensory defects, abnormalties of perception, difficulties in communication and social deprivations resulting from the handicap.

As a result of such problems, it is important that thorough evaluation of the child's speech and hearing mechanism and language development be made by a competent speech pathologist known to the pediatrician. Such records will also aid in the periodic re-evaluation of the child's progress, and the extent to which deficiencies have been overcome.

Another of the problems confronting the pediatrician is the attempt to classify the patient so that he may derive the fullest benefit from the educational program which the cerebral palsied child deserves. To enable the pediatrician to do this, the following flexible classification has been used, and cerebral palsied children grouped under one of the following five categories have derived greatest benefit (as a result) from their advisors:

(1) The cerebral palsied child needing custodial care—(a) those with severe mental deficiency who achieve only varying degrees of self-help; (b) those with severe mental deficiency, plus severe physical handicap, who achieve little; (c) those with severe physical disability but with little mental impairment, who make most progress intellectually in their environment.

(2) The cerebral palsied child with mental retardation—this group profits from an appropriate curriculum.

(3) The intelligent cerebral palsied child with severe or moder-

ate physical handicap—this group includes the child with alert mind who has perceptual, conceptual or behavioral disorders.

(4) The cerebral palsied child with mild physical handicap—so few in number that special schools are not needed.

(5) The non-motor handicapped child with brain damage—academic skill work will enable this group to participate partially in a regular school program.

These are but a few of the factors involved in the realistic management of the child with cerebral palsy. Pediatricians should serve two major functions in a program set forth to rehabilitate cerebral palsied patients. First, they must screen the population and select the handicapped children; secondly, they should treat the acute medical problems of the child in the program, and follow that child and note his progress. The pediatrician should remember, above all, to treat the child as a "whole," and this would include the cerebral palsied child's family. . . .

II. THE OBJECTIVES, PHILOSOPHY AND STRUCTURE OF REHABILITATION PROGRAMS FOR CHILDREN

WITH SPECIAL REFERENCE TO THE ROLE OF THE PEDIATRICIAN IN THE REHABILITATION AND PHYSICAL MEDICINE PROGRAM

A program of rehabilitation, if well organized, may offer advantages not only for children suffering from cerebral palsy, but also to those suffering from medical, neuromuscular and orthopedic conditions. The objectives of such programs are mainly three-fold:

(1) To overcome the original handicap with existing accepted techniques;

(2) To assist the child to live a full life by teaching him to accept and adjust to his physical limitations; the pediatrician will play a large role here in that he must minimize for the patient and his family the disease, while keeping in mind the extent to which the individual patient may be "pushed" while making adjustments;

(3) To promote public understanding and acceptance of the handicapped and the concept of "total rehabilitation."

The philosophy of the pediatrician, and those involved in re-

habilitation generally, should be based on the fact that the child with a physical handicap is first a child whose basic needs are those of any child. The pediatrician should remember that handicapped children are part of a family unit. The family must be included as participants in any program designed to aid their handicapped child.

Pediatricians serve to carry out the policies of a rehabilitation program by helping to diagnose the diseases involved, by evaluating the disabilities resulting therefrom, and by suggesting possibilities for help which are available for the disease condition. The pediatrician should know exactly which conditions require rehabilitation, and which need in-patient and out-patient care. For example, at the New York University-Bellevue Medical Center (Institute of Physical Medicine and Rehabilitation), the following diseases are given special rehabilitation:

(1) Neuromuscular conditions, such as poliomyelitis, cerebral palsy, muscular dystrophy, spina bifida and spinal cord lesions.

(2) Medical conditions, such as cardiac and arthritis conditions.

(3) Orthopedic conditions, including certain congenital deformities and amputations.

The pediatrician who suggests a rehabilitation program to any patient suffering from one or more of the above named conditions, should remember that follow-up and home care may be part of the task assigned to him later. At present, applications for service at New York University-Bellevue Medical Center are made by pediatricians, either by mail or telephone, so that the pediatrician may start treatment as soon as he thinks it is needed. Pediatricians serve especially to take detailed histories regarding cases needing rehabilitation. The pediatrician also examines the child in relation to the child's total health, development, and needs. It is interesting to note also that it is the duty of the pediatrician to supervise not only the medical or laboratory work-up in the rehabilitation program, but also the patient's diet, medication, consulting records, operative or emergency treatment, and recommendations to other physicians.

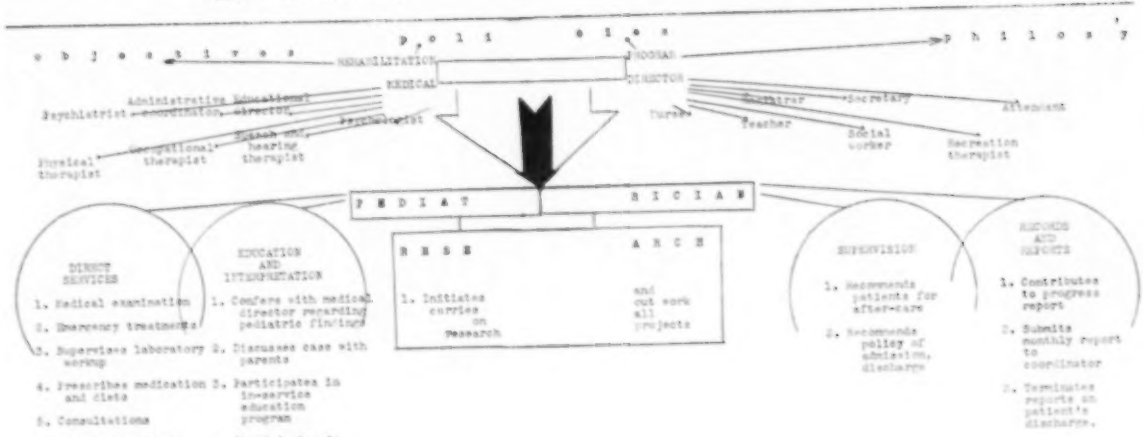
The structure of the program in rehabilitation may be stated simply, by referring to the individuals involved and the duties of each of these in the program. (There are, at present, very few

FIGURE I.

STRUCTURE OF A REHABILITATION PROGRAM, STATING SPECIFIC REQUIREMENTS ON PART OF THE PEDIATRICIAN IN SUCH A PROGRAM.

OBSERVE THAT MANY OF THE FUNCTIONS OF THE PEDIATRICIAN

MAY BE CARRIED OUT EVEN WITH THE INDIVIDUAL HOME CARE PATIENT.



programs in the United States that employ the necessary number of individuals). These are as follows:

(1) Pediatrician—who supervises all medical and laboratory work-up, and evaluates all records of the patient which are submitted by others in the program (Fig. 1).

(2) Physical therapist—measures range of motion of joints for contractures; gives muscle tests; evaluates child's abilities to perform the activities of daily living ("ADL").

(3) Nurse—evaluates child's ability to perform toilet and dressing ADL in bed and wheelchair.

(4) Occupational therapist—evaluates movements of arms in ADL.

(5) Speech pathologist—tests speech and hearing.

(6) Psychologist and psychiatrist—evaluate mental and emotional status of child.

(7) Ophthalmologist—tests visual acuity of child.

(8) Social worker—investigates social status of family.

(9) School teacher—makes educational evaluation.

(10) Recreational director—reports on recreational needs of child.

When each staff member completes his report, then the pediatrician, aided by a staff of physicians, suggests the rehabilitation possibility for the specific disability. The pediatrician is rapidly becoming the key figure in such a rehabilitation program involving disabled children. In Fig. 1, which shows the specific requirements of a pediatrician in a rehabilitation program, it will be noted that this phase of pediatrics requires considerable time on the part of the pediatrician; however, many of the duties listed may be delegated to other pediatricians in the program, so that practitioners may continue office duties while participating in the work.

III. CEREBRAL PALSY

Further Discussion Involving its Problems and Management Including Mention of Cases Handled by Pediatricians in the Recent Past and the Obligations of Pediatricians in Rehabilitation Programs

One of the first problems to be dealt with by the pediatrician is that involving the parents of the cerebral palsied child. In early

infancy, there is little noticeable difference between the cerebral palsied and non-cerebral palsied child. The pediatrician may be faced not only with the problem of treating the anxiety and tension of the parents, but also with relieving the feeling of guilt which some parents easily acquire when their child begins to show his handicap. The pediatrician who knows there are siblings in the family should see to it that the siblings receive the amount of care needed, in spite of the tendency on the part of some parents to spend all their time with the handicapped child.

By the time the cerebral palsied child is 2 to 3 years old, the diagnosis is well established. The child may be wearing braces or other special equipment. At this time, the pediatrician should be careful that the mother does not push the child to perform. The pediatrician also should take advantage, at this time, of the opportunity to show the parent how to participate directly in the child's rehabilitation by teaching him to use his appliances.

The time soon arrives when the child is eligible to attend school, if his mental state warrants this. At this time, the pediatrician is expected to help the parents relinquish the care of their child to others. Furthermore, the pediatrician must prepare families to give up their child temporarily, if such a child is to stay at a resident training center. The pediatrician may be faced with problems concerning the parents themselves, as for example, in the following case: "Carl, a young, severely handicapped athetoid, was brought to a hospital in childhood by cooperative parents. After some time, it became clear that Carl would never learn to walk. At this point, the parents are indifferent to the future care of their child, and make it clear that they wish him out of their home."

The pediatrician should remember, early in his relation with the family, that the cerebral palsied child shows slower psychological or biological development than normal children, and he should prepare the parents to meet this aspect of the disease.

Another pediatrician was faced with the case of Robert, who rarely left his house, because Robert's mother found it difficult to push his wheelchair. Playmates no longer visited the child, so that he began to "day-dream" and watch television. Robert slowly reached the point where he would not leave bed each morning, and he told his pediatrician that the lack of activity involved in his illness was driving him out of his mind. This is a true example

of a case which confronted a practicing pediatrician who, of necessity, found himself with some cerebral palsied patients.

The pediatrician should avail himself of knowledge concerning the care being given at present to severely handicapped youngsters. Too often, such children are pushed aside, and eventually they arrive at institutions which have little to offer. As soon as the pediatrician knows whether the child is of the pre-elementary school level, elementary school level, or post-elementary level, he should channel that child to the best group available for that child's care. This is possible only if the pediatrician is familiar with the specific condition from which the child suffers, plus the expectations he may have for that child's rehabilitation.

At present, even pediatricians, along with the lay public, are being awakened to the acute need for adequate care of handicapped children by the parents of these children. Some parents spend 24 hours a day with their children, and they are keenly aware of the additional training needed by present-day pediatricians in the art of physical medicine and rehabilitation.

There are 24 hospitals in New York City today which care for orthopedically handicapped children. The care which may be obtained in these hospitals varies from poor to excellent. In most instances, there are an adequate number of beds available for such children. Today, this is due to the recognized fact that the best place for the handicapped child is at home, and not in an institution. This includes also those patients who contract the usual minor medical problems which are suffered by all children.

One of the most important tasks of the pediatrician, in today's rehabilitation program, is to acquaint himself with the problems of under-institutionalization. At times, because a child has not spent enough time in an institution, he returns home with a greater disability than need be. Such under-institutionalization is usually due to the fact that the programming of such institutions is not as complete as it should be for the period of the patient's stay at that institution.

Pediatricians today tend to leave the care of the orthopedically handicapped child to the orthopedist or physiotrist. The pediatrician should participate actively with his collegues in this phase of medicine, so that full benefit is afforded the patient who is handicapped because of early brain injuries.

Probably the greatest advance made today in this field is the

cementing of relationships between the several specialties involved in the rehabilitation of the handicapped child. If each operated singly, the contribution to medicine would be minimal. Working together, there is recognition of the *psychosocial* needs of the patient, along with his medical needs, and the patient derives full benefit from the work.

One fact which the pediatrician should learn in his attempts to rehabilitate the disabled child is that muscles cannot be retrained or re-educated. However, the brain, which is the director of all bodily functions, can be retrained or re-educated. Therefore, the pediatrician should direct his energy in treating the handicapped child along lines which are commensurate with the patient's ability to be "re-trained." Only in this manner will the pediatrician attain the goal expected of him as an active participant in a pediatric rehabilitation program.

IV. SUMMARY

(1) The problems of the cerebral palsied child have been recognized since as early as 1853, but pediatric participation in rehabilitation programs which care for such patients is relatively new in the field of medicine.

(2) Medical research has revealed that cerebral palsied children present unique problems in development, learning and adjustment, and for these reasons, pediatric rehabilitation programs include a wide variety of workers, whose objective is to overcome a handicap with existing accepted techniques so that the patient may live a full life, and adjust to his physical limitations.

(3) The role of the pediatrician in a pediatric rehabilitation program is essentially three-fold: (a) To screen the general population for selection of handicapped children for participation in such a program. (b) To treat and follow the acute medical problems of the handicapped child. (c) To participate actively with others, especially the orthopedist, in the program of the rehabilitation team.

(4) The pediatrician should acquaint himself with facilities available for pediatric rehabilitation in and around his community, and he should help in the cementing of relationships between himself and the several specialties involved in pediatric rehabilitation.

(5) The pediatrician should remember to treat the patient who seeks rehabilitation as a "whole" in relation to his environment, and to his family.

ATYPICAL OSTEOGENESIS IMPERFECTA
LOBSTEIN'S DISEASE

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It is necessary, in the light of present information, to designate by the term osteogenesis imperfecta or ossium fragilitas a complex constitutional disorder of mesodermal origin.

The tendency to fracture is due to an osteoblastic defect in periosteal and endosteal bone formation leading to weakening of osseous structure. Since endochondral development of bone is unaffected, the linear process of growth is undisturbed.[1] The blueness of the sclerotics has been explained as due to an abnormal thinness of that layer leading to increased translucency. Bronson[2] has offered contradictory evidence. The sclera is of mesenchymal origin.

Otosclerosis is due to a sclerotic change in the ossicles resulting in interference with transmission of sound waves across the oval window.

Other changes which have been observed, but are not often mentioned, include laxity of joints, high, narrow, hard palate and extreme blueness of the tympanic membrane. The incidence of blue sclerotics is 50 per cent in families afflicted with the syndrome of osteogenesis imperfecta, while but one-third show the osseous defect. The noncongenital form is transmitted in the pattern of a Mendelian dominant. The age of the patients, described here, is such that otosclerosis (conceivably latent) is lacking; however, they all have the common added finding of amblyopia.

CASE REPORTS

M. L. F., female, age seven years, is a patient at the Muscatatuck State School and was admitted on July 11, 1952. She is blind. There are fracture deformities of the right clavicle and both tibiae. During May of 1954, she suffered a fracture of the right femur with apparently minimal trauma. Her height is 43 inches, weight, 40 pounds, head circumference (O.F.) is 19¾ inches. Serum calcium was found to be 13.5 mg. per cent and serum phosphorus 4.7 mg. per cent. X-ray study showed bowing of the right femur

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Fig. 1. Fracture deformity of femur with classically thin cortex and ground-glass-appearance of medullary portion.

Fig. 2. Showing marked generalized osteoporosis and thinness of cortex. The bowing of the femur is due to old fracture.

due to previous fractures. The skull films showed irregularity of ossification of the lambdoidal suture. Bone age was determined to be at the 4½-year-level. The patient's mental level was 6 years (Hays Binet). The ophthalmological consultant reported the presence of microphthalmos with occlusion of the right pupil and retinal detachment on the left. The sclerae were not blue. (Fig. 1 and 2).

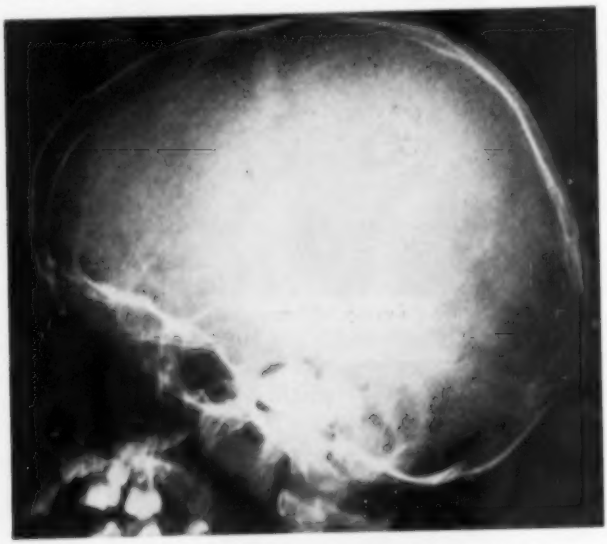
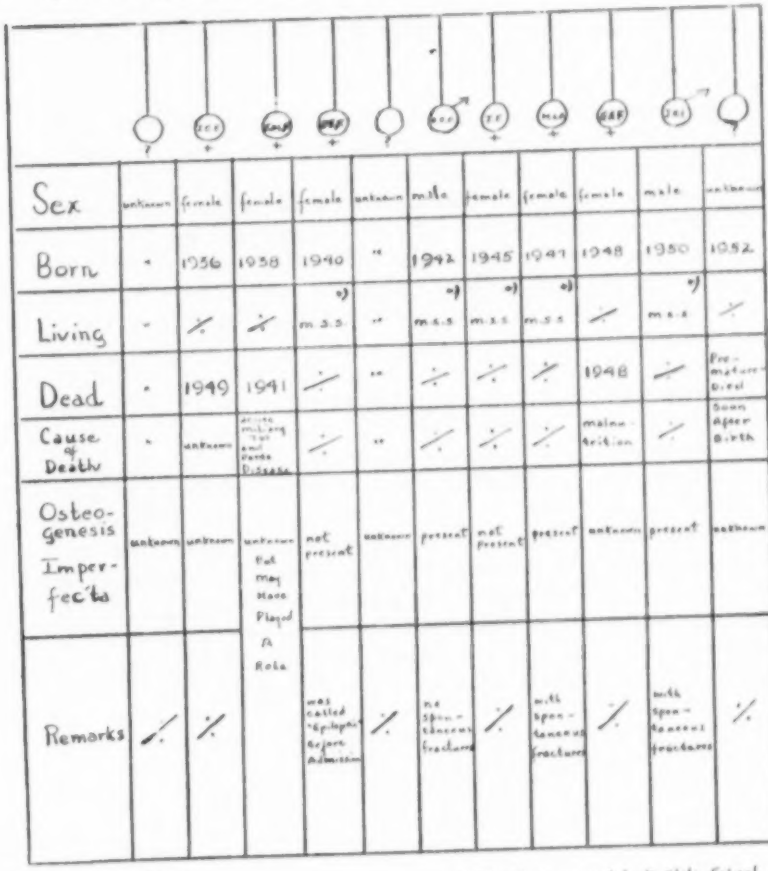


Fig. 3. Showing wormian bone formation in lambdoidal suture.

J. R. F., the younger brother, was admitted on July 11, 1952 at the age of four years. He suffered a spontaneous fracture of the left femur during 1954, and the healing process was rapid. His height was 36½ inches, weight 31 pounds, and the occipito frontal circumference of the head was 19 inches. The blood calcium was 15 mg. and the phosphorus was 4.2 mg. per cent. X-rays of the long bones showed classically thin cortex and generalized osteoporosis. Skull film showed wormian bone formation. (Fig. 3). This child was found to have bilateral microphthalmus and cataract.

E. A. F., the mother of the two preceding patients, was admitted on July 11, 1952, at which time she was 41 years of age. She had

had eleven children of whom only five survived. It was thought that her own father had sired all of her eleven offspring. This



woman was found to have an I.Q. of about 50. She stated that one of her uncles and a grandfather were inclined to fractures,

but this could not be verified. E. A. F. was 64 inches tall, weighed 163 pounds, and had an occipitofrontal circumference of 21 inches. Serum calcium was 16 mg. per cent and serum phosphorus was 3.1 mg. per cent.

DISCUSSION

The diagnosis of atypical osteogenesis imperfecta is felt justified. (Fig. 4). The blue sclerotics are absent and the possible latence of otoscelerosis is indeterminate.

The fragility of bones was noted when spontaneous fracture was found to develop in the two youngest family members. One other sibling, twelve years of age, showed radiologic changes consistent with the diagnosis of osteogenesis imperfecta, but had never suffered a fracture. These changes included generalized osteoporosis of long bones, thinness of the cortex of long bones, and wormian bone formation in the lambdoidal sutures. This child is the only living male descendant, except for the youngest child, who has been discussed above. The condition does not appear to be sex-linked as the most severely afflicted is a female. The factor of incest, in the mechanism of transmission of the defect, requires further study.

The author wishes to acknowledge the cooperation of Drs. H. F. Sudranski, H. E. Stadler and H. E. Rothring, consultants, and Dr. H. Leland, Director of Psychological Service. Mr. J. Calandro was responsible for the laboratory data and the roentgenograms. Dr. Abraham Levinson, Chief of Staff, Children's Division, Cook County Hospital, Chicago, Illinois, gave invaluable advice.

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CORTISONE THERAPY IN SYDENHAM'S CHOREA

REPORT OF CASE

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Introduction. In 1685, Sydenham described a disorder characterized by irregular, rapid, abrupt, purposeless movements and called it "Saint Vitus Dance" in recognition of the fact that sufferers from these choreic "dancing" movements prayed to St. Vitus for intervention. Later the disease was named for its describer—Sydenham's chorea.

Although chorea minor is usually considered as a manifestation (major) of rheumatic fever, there have been cases similar in all respects to a choreic attack reported as a complication in scarletina, pneumonia and measles. There have also been several cases of acute chorea reported in association with pregnancy, representing either a reactivation of an old case, or, in some instances, an initial attack.[1] The relationship of chorea to rheumatic fever has often been questioned because of the low incidence of rheumatic heart disease in these patients. Over 75 per cent of all cases of chorea, however, are either preceded or followed by other rheumatic fever manifestations, the "pure" case representing the exception rather than the rule.[2]

At the present time the etiology of Sydenham's chorea is not known, although there are many theories, most of which are variations of two basic mechanisms. In the first, the possibility of a variety of hemolytic streptococcus which produces a toxin acting specifically upon the central nervous system, particularly the basal ganglia, is entertained. While this theory would be helpful in explaining why all rheumatic fever victims do not exhibit chorea, i.e., a different variety being the etiological factor, no such toxin has been isolated or demonstrated. The second mechanism, which is more generally accepted, is that chorea is merely another expression of the antigen-antibody reaction believed to be involved in the rheumatic fever process. This concept of the disease receives support from post-mortem findings of proliferative changes in the intima of the medium sized blood vessels of the brain which are the same as those found in other organs of those with rheumatic fever.[1]

Acute chorea is found, almost exclusively, in the five to fifteen year age group, with a maximum incidence at eight years. Females are affected three times more frequently than males and, unlike

Huntington's chorea, a hereditary transmission has not been demonstrated. The attacks, which tend to recur and are often prolonged, usually end in complete recovery. Most frequently they consist of a triad of emotional instability, purposeless movements and muscular weakness.[1, 2]

Therapy. In the past the therapy of chorea has revolved about the removal of sensory and emotional stimuli which tend to increase the purposeless movements, e.g., bed rest, isolation, quiet and sedation, fever therapy in the more severe cases as it appears to shorten the course of the disease slightly, and other general measures, such as warm baths and packs, psychotherapy, and the use of barbiturates, chloral and other sedatives. With the advent of cortisone and ACTH, and the concept of their roles in stress and antigen-antibody reactions of the body[4], these drugs were tried in rheumatic fever with the idea that they might interfere with the antigen-antibody reaction believed to be responsible for the Aschoff body formation and resultant damage of the disease. Since that time, many series have been run on rheumatic fever patients, most of which have yielded fairly encouraging results. In one series[5], eleven cases of rheumatic fever were given ACTH (30-100 mgm. q6-8h for three days, then 40-60 mgm. for three days). Of the six cases representing initial attacks, two showed no residual cardiac damage, while three showed only equivocal evidence of mild cardiac damage. One case was not counted since the drug was withdrawn due to side effects. Of the remaining five cases with recurrent attacks, none showed any evidence of increased cardiac damage over that already present. Other series have shown similar results.[6, 7] On the whole, the effect of cortisone and ACTH is reflected by a return of the erythrocyte sedimentation rate to lower or normal levels, a return of the electrocardiogram (EKG) to normal limits, an improved clinical picture and the prevention of residual cardiac damage. Further evaluation of these drugs will be necessary, however, before any definite conclusions may be reached.

Regarding chorea as a manifestation of rheumatic fever, it is not surprising that cortisone (ACTH) should be evaluated in this disease. Possibly the first attempts at cortisone therapy in chorea, occurred with the use of fever therapy, since this probably acted as a stress,[8] raising the level of adrenal secretion. So far, not many cases have been reported and the results have been conflicting. In

one series[9] two cases were given daily doses of cortisone, beginning with 100 mgm. for three days, then 50 mgm. for six days, then 75 mgm. for five days. Clinical evaluation revealed no change at the end of this regime. Other results have shown shortening of the course of the attacks.[10] More extensive series must be studied under more standardized conditions of dosage, and evaluation will be necessary before an estimate of the efficacy of this type of therapy in chorea can be determined.

CASE REPORT

The following case is presented as an example of what appears to be a dramatic effect of cortisone in shortening the course of a choreic attack, with the full realization that such isolated accounts, under poorly controlled conditions, have little meaning in evaluation of cortisone therapy.

B. R., an eight-year-old Puerto Rican female, was admitted to the hospital on March 10, 1954 with a complaint of purposeless hand movements, rolling of the eyeballs, grimacing and difficulty in writing and walking, all of six days' duration. There was a history of an upper respiratory infection three months prior to admission, but no symptoms referable to rheumatic fever or a glomerulonephritis could be elicited. This was the first attack of this nature, with no history of convulsive seizures, epilepsy or other central nervous system disorders. The family history was noncontributory, with no evidence of Huntington's chorea, or other neurological diseases in the family. Aside from the above mentioned movements, physical examination revealed no signs of a focus of infection in the ears, nose or throat. The heart showed no enlargement to percussion, a sinus arrythmia, P_2 greater than A_2, no thrills or murmurs, and a ventricular rate of 100. There was no pulse deficit. On neurological examination the deep tendon reflexes were found to be equal and active with a typical "hung-up" patellar reflex. The superficial abdominal reflex was present and active, the pupils reacted to light, and no Babinski or Hoffman reflex was elicited. No muscle weakness was noted in the strength of hand grasp, but no more accurate evaluation was made. The child's emotional status did not seem abnormal, but no formal evaluation was made. The child was well oriented. A hard, round, nodular mass was palpated just posterior to the medial malleolus of the left ankle,

and was found to be freely movable. Laboratory data at this time revealed the following:

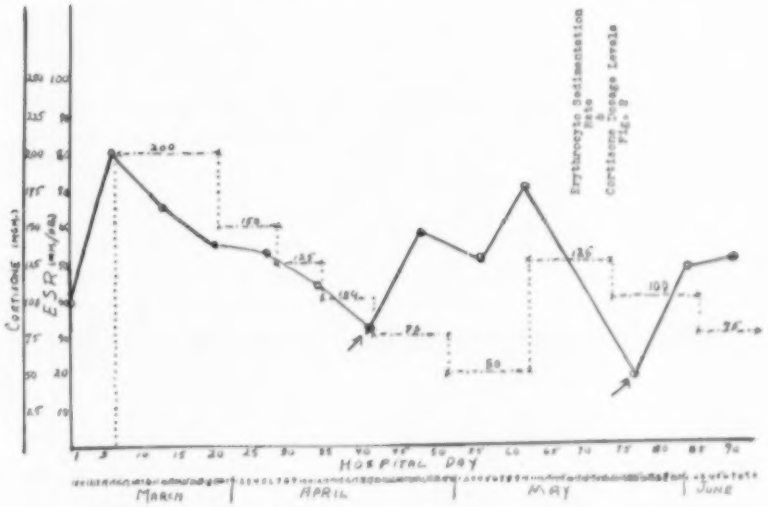
ESR (Cutler): 3/40
RBC: 5,010,000
Hb.: 12.5 gm.
WBC: 8,050 with 56 per cent segmented polys, 3 per cent staffs, 38 per cent lymphocytes, 3 per cent eosinophils.
Chest X-ray: No cardiac enlargement
EKG: Sinus arrythmia with no increased P-R interval or other abnormality.

The admission diagnosis was Sydenham's chorea.

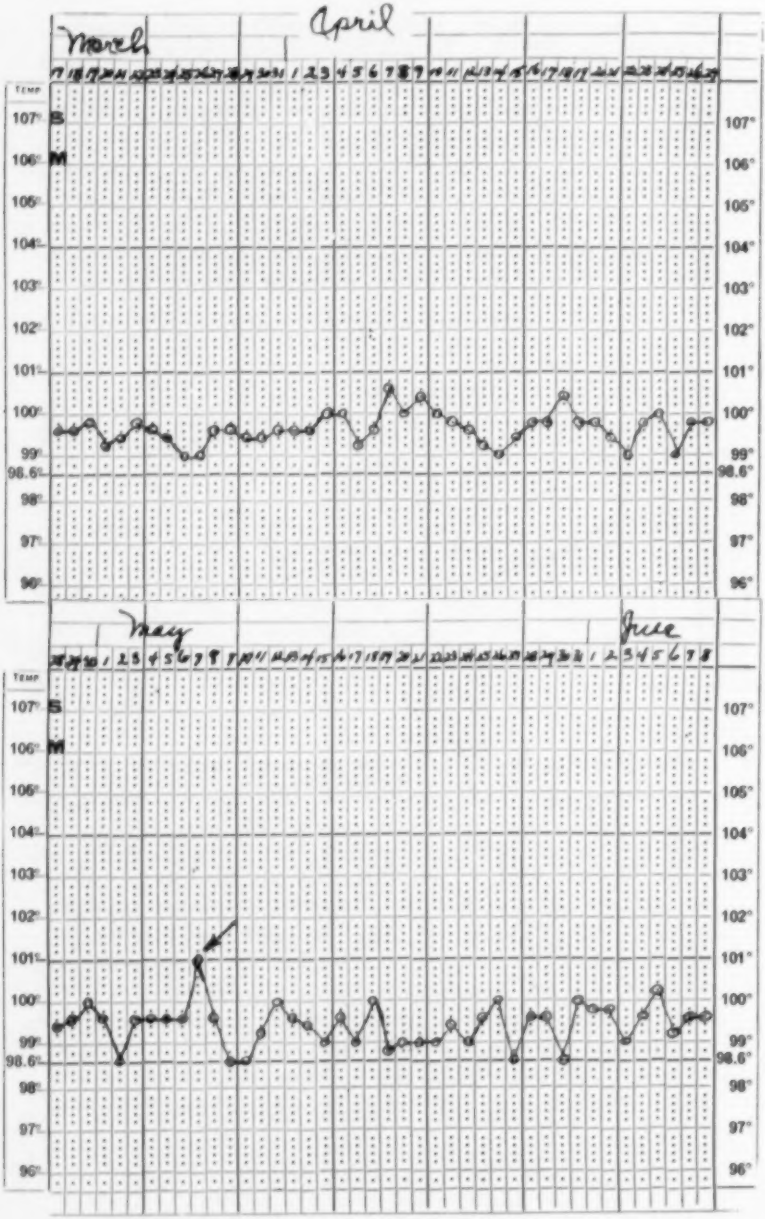
				WBC					
DATE	ESR	RBC	Hb	TOTAL	P	L	M	E	B
3/10	3/10	5,010,000	12.5	8,050	59	38	—	3	—
3/15	10/80		—	13,300	65	30	2	5	—
3/22	15/65		—	15,600	82	13	3	3	—
3/29	8/55		—	12,600	69	25	—	6	—
4/5	6/53		—	10,300	74	24	—	2	—
4/12	4/44		—	13,650	68	25	3	4	—
4/19	4/38		—	10,150	63	34	—	3	—
4/26	8/58		—	12,800					—
5/4	7/52		—	9,950	52	32	2	4	—
5/10	8/70		—	11,550	63	30	2	5	—
5/25	3/29		—	15,900	65	29	3	3	—
6/1	7/48		—	10,900	53	37	5	3	2
6/7	9/50		—	8,650	68	27	3	2	—

Hospital Course. The patient was placed on bed rest and followed with sedimentation rates and blood counts. (See Fig. 1.) On the sixth hospital day a course of cortisone was started, the initial dosage of 200 mgm. being reduced by 25 mgm. about every ten days. (See Fig. 2). At this time prophylactic dosages of 900,000 units of Bicillin were started daily. On the twelfth hospital day (sixth cortisone day) all clinical evidence of the choreic movements were absent, making the total length of the attack (clinical) eighteen days. On admission and during the entire hospital course, the only febrile episode occurred on May 7, 1954, when a mild upper respiratory infection occurred. (Temperature: 101° F.).

This disappeared rapidly when treated with procaine penicillin (600,000 units). A moon facies was noted soon after the onset of the cortisone therapy and is still present, but there has been no evidence of increased water retention or systolic hypertension (pressure has never risen over 120 mm. Hg.). KCl (gr. xv) has been given daily with the cortisone to minimize potassium loss. The ESR showed a steady decline from a peak of 80 mm. per hour (March 15, 1954), to 32 mm. per hour (April 19, 1954). A second elevation of the ESR began at this time, reaching another peak at 70 mm. per hour (May 10, 1954). A dosage level of 50 mgm. of



cortisone was being given at this time. This was again increased to the 125 mgm. level and the ESR again fell, this time to within normal limits (20 mm. per hour). Following two successive drops in dosage to the 75 mgm. level, the ESR again rose, reaching the present level of 50 mm. per hour (See Fig. 2). So far, with the exception of a soft functional murmur over the pulmonic area heard on one occasion, there has been neither clinical nor electrocardiographic evidence of a heart lesion. The nodule felt on the left ankle at the time of admission is still present. At the present time, it is thought to be a fibroma rather than a rheumatic nodule.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Stool and urine studies have failed to show any evidence of parasites or urinary tract infection. Studies of the paranasal sinuses and a urine culture are still pending at this time. Physical examination at the present time is essentially negative. The child is still hospitalized and under cortisone therapy (75 mgm. per day).

DISCUSSION

The total duration of the choreiform movements in this case was eighteen days, the last six of which the patient was on cortisone therapy. Since the references consulted[1, 11, 12] give the usual duration of an attack before spontaneous recovery as six to eight weeks, it is felt that the cortisone therapy was responsible for the early remission of symptoms.

At present, based on our knowledge of the etiology of rheumatic fever and chorea, the mechanism of action of the adrenal corticosteroids, and the principles underyling antigen-antibody reactions in general, any discussion of the effects of cortisone on chorea must be on a highly theoretical basis. One rationale for the use of cortisone (or ACTH) in chorea or rheumatic fever is based on the assumption that both are manifestations of a bacterial allergy (*S. hemolyticus*) and that the cortisone acts by blocking the antigen-antibody reaction involved. If this assumption is accepted, then the sedimentation rate can be interpreted as a rough reflection of the rate at which the allergic process is progressing, since it is determined primarily by the proportions of fibrinogen, albumen and globulin in the blood. Thus, if the cortisone blocks the antigen produced by the streptococcus, antibody formation will decrease, the gamma globulin fraction of the blood associated with antibody formation will decrease, and this would cause a similar decrease in the ESR . This is, naturally, dependent upon the absence of factors which would increase the albumin and fiberinogen factors (e.g. infection).

In the graph (Fig. 2) of the E.S.R. and cortisone dosage levels, it may be noted that the elevations of the E.S.R. occur on the lower dosage levels and are counteracted by increasing the dosage. It is felt, therefore, that these "escapes" of the E.S.R. are due to inadequate blood levels of cortisone, either from inadequate dosage, adrenal gland suppression, or both.

Attempts at gauging effective dosage levels of cortisone and ACTH by means of the eosinophil count have been studies by Thorn[13] and Randolph.[14] The average eosinophil count in either child or adult is of the order of 200 cells per cubic millimeter. If the eosinophil count is taken four hours following the morning dose of cortisone for the first three or four days of therapy, the eosinophil decrease is usually found to be of the order of 50 per cent. The validity of this test as an indication of adequacy of dosage, however, has been challenged. In one series [15] in Canada involving fifty-six cases composed of rheumatoid arthritics and those suffering from bronchial asthma, it was found that the majority of patients treated showed clinical improvement with or without a decrease in eosinophils. This same study found that the esoinophil count is also subject to such a wide normal variation (30 to 250 cells/mm.^{3}), that the clinical rule that a count of less than 30 eosinophils per cubic millimeter is indicative of adequate dosage levels is a questionable one.

Uric acid—creatinine ratios and urinary excretion of 17 ketosteroids have also been employed in the evaluation of dosage levels. However, the former is not a very accurate measure and is subject to many variations from individual to individual, and the latter requires too much time and skill to make it anything but a research tool, although it is an accurate measure.

It is interesting to note that ACTH has been shown[15] to be a stronger eosinopenic agent than cortisone. Also, if cortisone is administered at a fixed dosage level for a period of three to twenty-three days, the eosinophil count begins to "escape" or rise, but this will not occur when ACTH is used. Moreover, it has been observed that if a second course of cortisone is begun shortly after the first, the eosinophil decrease caused by the second course will be less than the first. Since the escape phenomenon does not occur with ACTH, it would seem that it represents a suppression of the adrenal cortex by increased cortisone blood levels. This may also account for the escape of the sedimentation rate noted in the above case history.

Since administration of ACTH would theoretically suppress the pituitary by producing high cortisone levels, it is necessary to postulate that the pituitary depressant effect of ACTH working through the adrenal is much less effective than direct inhibition of

the pituitary by the adrenocortical hormone. This could be explained by assuming that blood levels of ACTH reached during therapy drive the adrenal cortex at a maximal rate and that the ACTH derived from pituitary secretion during ACTH therapy plays no role in determining adrenal output. The exact mechanism of the escape phenomenon is, however, not known.

Because ACTH has been shown to exhibit no decrease in eosinopenic effect, it would appear that its use might prove very helpful in maintaining high cortisone levels in chorea when used alone or in conjunction with cortisone. By minimizing the factor of adrenal suppression, the dosage levels in the blood would also tend to be more stable. In one series[10], ACTH and cortisone were given in ratios ranging from 1:1 to 1:3 every six hours to six children between the ages of seven and thirteen with chorea minor. It appeared that maximum benefit occurred as the ratios approached 1:1 and that a combination of the two drugs was more effective in causing a cessation of symptoms than was reported in other series in which the drugs were used alone. The results on so few cases were not felt to be considered conclusive.

As noted above, the case of "pure" chorea is not very common. Since, in the case presented, the sedimentation rate has remained elevated over a long period of time after subsidence of symptoms and the white blood count reveals a typical leucocytosis (8,000 to 25,000 cells/mm.^{3}) with a predominance of neutrophils (Fig. 1), it is thought that there has been an underlying rheumatic process occurring. It is not unreasonable to assume that the cortisone therapy has suppressed other rheumatic manifestations such as a carditis or a migratory arthritis.

SUMMARY

This case was presented as an example of a fairly rapid remission of clinical manifestations of Sydenham's chorea under cortisone therapy. It is felt that aside from relief of symptoms, cortisone also acts to suppress further damage from an underlying rheumatic process.

There still appears to be a need for some laboratory test which will reflect the adequacy of dosage levels, in ACTH and/or cortisone therapy, with speed and accuracy, by a reasonably simple technique.

In order to determine the role of the adrenal hormones in chorea, it will be necessary to have many more series compiled under more standardized conditions of dosage levels. The use of combinations of ACTH and cortisone should be evaluated further.

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OXYGEN AND RETROLENTAL FIBROPLASIA: A SEVEN YEAR SURVEY. (Lancet, 2:258-260, Aug. 7, 1954). The relationship between the use of high oxygen concentrations in the treatment of premature babies and the development of retrolental fibroplasia has been widely discussed. This paper records the disappearance of the disease from two nurseries for premature infants in Manchester and relates this change to restriction in the use of oxygen. During the past seven years 83 cases of retrolental fibroplasia occurred in these two nurseries. Thirty-six of these children have permanent changes, and 21 are blind. Since October, 1951, the amount of oxygen used for each infant has been severely restricted. Since November, 1951, there has been no case of blindness due to retrolental fibroplasia. The incidence of incomplete and temporary changes has been similarly reduced. These observations confirm the important role that oxygen plays in transforming a rare sporadic disease into a major cause of blindness. The authors are satisfied that the elimination of the disease by restriction in the use of oxygen can be achieved without detriment to the survival rate.—*J.A.M.A.*

THE USE OF CHLORPROMAZINE AS AN ANTIEMETIC IN CHILDREN

LOUIS A. WIKLER, M.D.
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Although several reports have appeared on the use of chlorpromazine (Thorazine®) in the treatment of nausea and vomiting in adults[1, 2, 3], none have yet appeared on this use in children. There are undoubtedly several reasons for this, not the least of which is the fact that vomiting in children rarely is prolonged. Moreover, chlorpromazine, until recently, was not available even for investigational use in dosage forms especially suitable for children.

Still, vomiting can be alarmingly persistent in such infectious states as gastroenteritis, acute tonsillitis and various viral infections; the loss of water and electrolytes weakens the child and retards recovery. Moreover, the parents become exceedingly distraught when they find that the child cannot retain even fluids. Consequently, it seemed worthwhile, when chlorpromazine was made available for investigational use in syrup and suppository form, to evaluate its usefulness as an antiemetic in children.

MATERIAL AND METHOD

This series consisted of forty-eight children with vomiting secondary to acute gastroenteritis, tonsillitis and nonspecific infections presumably of viral origin. All patients were seen in the office or in their homes. Table 1 indicates the number of children in each diagnostic group, as well as the age ranges.

TABLE 1.

Diagnosis	*No. of Children*	*Age Range*
Acute gastroenteritis	15	3 months to 8 years
Tonsillitis	11	2 to 8 years
Virus infection	21	1½ to 12 years
Roseola	1	2 years
	48	3 months to 12 years

The parents of these children, when told of the proposed evaluation, agreed to cooperate by reporting later whether the drug helped relieve their child's distress. Each was given a supply of chlorpromazine, in syrup or suppository form, and asked to keep

a careful record of the number of doses administered and to note when the child ceased vomiting and was able to retain fluids and solids. When these data were collected, they were compared with a typical patient response established in children, selected at random, who had received no chlorpromazine. Without such references, interpreting data on such a series would be meaningless because of the self-limiting nature of these illnesses. Specific therapy, including antibiotics and sulfa drugs, of course, was given when indicated. The testing of throat smears and stool samples was deemed unnecessary.

Thirty-nine of the children were treated with suppositories; nine with syrup.* In one child, who developed diarrhea, it was necessary to shift from the suppository to syrup on the second day of treatment. Children under six usually received the 25 mg. suppository, while those over six were given a 50 mg. suppository made by cutting a 100 mg. suppository in half. The usual dosage of syrup was one teaspoonful (10 mg.) t.i.d. or q.i.d. The parents were instructed to continue the medication until the symptoms of nausea and vomiting were relieved. Thirty-four of the forty-eight children received penicillin, tetracycline, sulfas and salycilates in conjunction with chlorpromazine.

RESULTS AND DISCUSSION

The nausea and vomiting accompanying gastroenteritis, tonsillitis and other infections were controlled to an impressive degree. In nineteen of the thirty-nine children treated with suppositories, one application sufficed to stop vomiting. Most of the remaining children, including the nine treated with syrup, were relieved of nausea and vomiting within the first day of treatment, that is, on a dosage schedule of suppositories twice daily or syrup (10 mg./5 cc.) three or four times daily. And, although their child was asymptomatic, some of the parents continued the medication throughout the second day. Some continued it until the third day, explaining that it was "just for good measure."

In two of the forty-eight patients results with chlorpromazine were unsuccessful. One, in whom vomiting was arrested, continued to complain of nausea. In the other, vomiting persisted,

*The suppository and syrup forms of chlorpromazine (Thorazine®) were supplied for investigational use by Smith, Kline & French Laboratories, Philadelphia, Pa.

although the bouts were not as frequent. It is felt that in this last case the situation was complicated by spasmodic coughing.

In children who did not receive chlorpromazine, nausea and vomiting frequently persisted for as long as 48 hours, sometimes longer. And while therapy with antibiotics and sulfas may have contributed to nausea and vomiting in some children, it is doubtful that it contributed appreciably since most of the children had received these drugs previously and tolerated them.

Drowsiness was noted to some degree in nearly half of the patients. Ordinarily, the sedation would be considered a side-effect, but in these cases it was beneficial. No untoward reactions were observed.

CONCLUSIONS AND SUMMARY

1. Chlorpromazine (Thorazine®) in syrup and suppository forms was administered to forty-eight children suffering from nausea and vomiting secondary to gastroenteritis, tonsillitis and various infections presumably of viral origin.

2. Results of treatment, compared to that in untreated children, indicate that chlorpromazine is a highly effective antiemetic and is safe for use in children.

3. Drowsiness, occurring in 42 per cent of the children, was the only side effect noted. It was considered a definite therapeutic advantage in treating children for the ailments described.

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DEPARTMENT OF ABSTRACTS

KIESEWETTER, W. B.; NELSON, P. K.; PALLADINO, V. S. and KOOP, C. E.: NEONATAL TORTICOLLIS. (Journal American Medical Association, 157: 1281, April 9. 1955).

In a series of 32 infants and 14 older children with congenital sternocleidomastoid tumefactions and/or torticollis, there was a high incidence of obstetric abnormalities. It is felt that these were the result of a prenatal lesion and not the causative factor in the torticollis. Surgical resection, particularly when done early in life, can be expected to produce good results in practically all cases.

AUTHOR'S SUMMARY.

JANEWAY, C. A.: PEDIATRIC RESEARCH IN THE AGE OF THE COMMON MAN AND THE EXCEPTIONAL CHILD. (Journal American Medical Association, 157: 1289, April 9, 1955).

If this talk has any message, it is a plea for the whole, for the recognition that research is part and parcel of the total academic medical enterprise. To the extent that this new type of support for what medical schools are doing undermines the basic academic principles of tenure and freedom, to the extent that it forces medical research into channels where lay people think it ought to go rather than allows it to flow where the ground swell of basic scientific advance and the imagination of free and creative investigators takes it, it can hurt medical education and the very cause for which the funds were obtained, the promotion of health and the conquest of disease. I do not personally believe that this is necessary or inevitable, but rather that, with sufficient thought and effort on the part of those concerned with university administration on the one hand, and those involved either as administrative officers or as advisors to the foundations and the governmental agencies on the other, policies and practices can be evolved to make the wisest use of the extraordinary vitality and energy that has created these vigorous new institutions.

AUTHOR'S SUMMARY.

SCHENCK, H. AND LEIDER, S.: HERNIA AS A CAUSE OF COLIC IN INFANCY. (New York State Journal of Medicine, 55:1467, May 15, 1955).

1. The term colic denotes a symptom caused by paroxysmal abdominal pain. Many reasons for colic have been cited, both functional and organic. 2. Among the organic causes appendicitis and intussusception are invariably mentioned, whereas inguinal hernia is almost never listed as an etiologic factor. 3. Eleven cases are reported which illustrate that inguinal hernia may cause colic in infancy. The colic was produced by repeated bouts of incarceration, and the symptoms in every case ceased and did not recur following surgical repair of the hernia. 4. In investigating the cause of colic in infants, inguinal hernia should always be borne in mind. When present, the hernia should be repaired surgically since the attacks of colic may be due to periodic incarceration. 5. One should not be satisfied with one or two negative examinations for hernia in colicky babies. Mothers should be alerted to the possibility of this condition as a cause of colic, and their observations should be respected, since they are generally excellent observers. 6. In enumerating the causes of colic in infants more emphasis should be given to inguinal hernia as an organic cause since this condition is far more common in early infancy than is either appendicitis or intussusception. Furthermore, inguinal hernias become incarcerated much more frequently in early infancy, at which time the incidence of "idiopathic colic" and "unexplained crying" in babies is highest. 7. The morbidity and mortality resulting from surgical repair of inguinal hernias in infancy is minimal. All of our cases left the hospital on the day following operation, and the course was uneventful in each instance. 8. By constantly isolating and correcting definite causes for colic in infancy, it is hoped that the term "idiopathic" will be lowered to an irreducible minimum.

AUTHOR'S SUMMARY.

ILLINGWORTH, R. S.; PHILPOTT, M. G. AND RENDLE-SHORT, J.: A CONTROLLED INVESTIGATION OF THE EFFECT OF DIET ON ACUTE NEPHRITIS. (Archives Disease in Childhood, *29*:551, Dec. 1954).

Forty-two children, aged from 2 to 14 years, suffering from acute nephritis, were allocated by the random sampling method to one of two dietary regimes, severe protein restriction and the normal ward diet. No case was observed for less than a year; the average period of observation was over 2½ years. The two groups were

shown to be statistically comparable. Strict criteria for "healing" were laid down. There was one death in the low-protein group. In the remaining children no significant difference in the rate of healing was observed. We concluded that we had been unable to demonstrate any advantage in protein restriction.

AUTHORS' SUMMARY.

STEINBACH, H. L.; ROSENBERG, R. H.; GROSSMAN, M. AND NELSON, T. L.: THE POTENTIAL HAZARD OF ENEMAS IN PATIENTS WITH HIRSCHSPRUNG'S DISEASE. (Radiology, 64:45, Jan. 1955).

Severe reactions and deaths have occurred not infrequently in patients with Hirschsprung's disease during or immediately following administration of an enema. The cause of these reactions has not been conclusively demonstrated, but some evidence has been provided to suggest the possibility of water intoxication due to rapid absorption of water from the colon. It is also possible that reflex cardiac inhibition and shock may be produced by abnormal distention of the colon. It is recommended that isitonic saline solution be used in enema studies in patients in whom Hirschsprung's disease is suspected. It is also recommended that the smallest amount of solution necessary be introduced under low pressure, and that as much as possible of the fluid be recovered after the procedure has been completed. Five cases are reported.

AUTHORS' SUMMARY.

GREENBERG, M.; APPLEBAUM, E.; PELLITTERI, O. AND EISENSTEIN, D. T.: MEASLES ENCEPHALITIS. TREATMENT WITH GAMMA GLOBULIN. (Journal Pediatrics, 46:648, June 1955).

(1) Over a period of 5½ years there were observed 159 patients with measles encephalitis under the age of 15 years. Of these, 51 were treated with gamma globulin and 108 were not. (2) About 25 per cent of the children died or developed permanent sequelae, and the percentage was the same in the two groups. (3) No significant differences in results between the treated and the untreated were found when comparisons were made by age groups or by amount of treatment or by severity of case, irrespective of whether the difference was based on deaths or sequelae or both. (4) Gamma globulin has no value in the treatment of measles encephalitis.

AUTHORS' SUMMARY.

MASLANSKY, E. AND JOLLIFFE, N.: FACTORS RELATED TO UNDERWEIGHT IN A SELECTED GROUP OF 100 CHILDREN IN NEW YORK CITY. (American Journal Public Health, 45:1054, Aug. 1955).

We have reported on our experience with a group of 100 underweight children whose growth patterns were studied at the nutrition clinic. We found this physical syndrome constituted a problem to the mother, child and community regardless of its clinical significance. Diet education alone, in the conventional sense, could not resolve the problem. Indeed, it often heightened the mother's anxiety about her underweight child. A search for more effective ways of dealing with the problem led to the exploration of some of the conditioning factors. Ignorance and economic pressures undoubtedly were involved, but it is difficult to evaluate to what extent, for often they were complicated by physical and social ills that impaired the mother's capacity to function adequately. What emerges most clearly from our study is that most of these children presented eating problems that began in infancy or early childhood and were rooted in the social, cultural and psychological complexities of their environment. A better understanding of the individual, the family setting, and the factors that motivate eating behavior will stimulate the development of new approaches to the problems of nutrition education, to the end of improving our services to those who seek our help. AUTHORS' SUMMARY.

CHUSID, E. AND LAPIN, J. H.: SIGMOIDOSCOPY IN THE DIAGNOSIS OF AMEBIASIS AND ULCERATIVE COLITIS IN INFANTS AND CHILDREN. (New York State Journal Medicine, 55:2195, Aug. 1, 1955).

Four cases of amebiasis and three cases of ulcerative colitis in infants and children have been presented, in which the diagnosis was not established until sigmoidoscopy with examination of the aspirated mucosal exudate was performed. These 7 cases did not present diarrhea as the chief complaint, but instead there were such diverse symptoms as developmental failure, arthralgia, epistaxis, vomiting, abdominal pain, and fever of unknown etiology. It is suggested that sigmoidoscopy is an exceedingly useful aid in the diagnosis of these two conditions and that it presents no danger to the infant or child when performed carefully and skillfully.

AUTHORS' SUMMARY.

BOOK REVIEWS

ADVANCES IN PEDIATRICS. Volume VII. Edited by S. Z. Levine, M.D. Cloth. Pp. 351. Illustrated. Price $8.00. Chicago; Year Book Publishers, 1955.

This present volume is the newest addition to an excellent series of books presenting concise and yet complete reviews of timely subjects. This seventh volume contains seven articles: Fibrous Defects in Cortical Walls of Growing Tubular Bones, by John Caffey; The Urinary Tract in Childhood, by Meredith F. Campbell; Malnutrition in Infancy and Childhood, with Special Reference to Kwashiorkor, by Federico Gómez et al.; Phonocardiography in Children, by Edgar Mannheimer; Infantile Cerebral Palsy, by Meyer A. Perlstein; Mucoviscidosis, by Harry Shwachman and Hugo Leubner; Congenital Magacolon, by Orvar Swenson. The subjects of Kwashiorkor, Cerebral Palsy and Muscoviscidosis are receiving increasing attention lately and these reviews make excellent additions to the recent literature.

MICHAEL A. BRESCIA, M.D.

TRIUMPH OF LOVE. By Leona S. Bruckner. Cloth. Pp. 213. Price $3.00. New York: Simon and Schuster, 1954.

Mrs. Bruckner has written a book recounting her problems and experiences as a result of giving birth to a handicapped child, the handicap consisting of a congenital absence of practically both arms. The book is simply written yet is replete with emotional impact. The emotional impact of the birth of such a child to the parents and the medical attendants seems almost insurmountable. The story of rejection and finally total acceptance of the child by the parents makes a gripping story. How to tell a new mother that she has delivered a handicapped child, regardless of the type of handicap, taxes any medical attendant's ingenuity. The choice of words and the attitude of the attending physician are most important. I wonder if the use of the word "monstrosity" in describing the child to the mother soon after birth was wise; also trying to dispatch the newborn with the aid of the father, while keeping the mother in the dark as to the true state of

affairs, was the best course to adopt. I feel that these two points added immeasurably to the mental anguish and turmoil of these parents. How these parents met their problem shows that most human beings, when put to the test, can bring out their best qualities and become dedicated beyond their own immediate problem. The particular problems which arise in caring for and rearing a child without arms are of great value for the phsyician who might need this information to guide and soothe some other parents. This book is of value for the physician and for the general public since not the least of the troubles of the parents of a handicapped child is the inconsiderate and ignorant attitude of the neighbors. Mrs. Bruckner is to be commended for writing of her experiences with such a child. MICHAEL A. BRESCIA, M.D.

FIBROCYSTIC DISEASE OF THE PANCREAS. A Congenital Disorder of Mucus Production—Mucosis. By Martin Bodian, M.D. Cloth. Illustrated. Pp. 244. Price $9.50. New York: Grune & Stratton, Inc., 1953.

The meat of this monograph is a pathological study of a most interesting disease which is receiving lately a great deal of attention. This volume also contains a brief protocol of the 116 cases on which the author bases his discussion and conclusions. Many names have been suggested for this condition — most recently mucovisidosis and the author's suggestion of mucosis because of the essential pathology which is a disturbance of the mucus secreting cells not only of the pancreas and the lungs but also of the salivary glands and gastro-intestinal tract. In the final chapter the author discusses briefly pancreatic dysplasias which differ essentially from the mucosis in that the pathology is limited to the pancreas as a pancreatic disease only and not a disturbance of the mucus secreting glands of which the pancreas happens to be one. This is a valuable book. MICHAEL A. BRESCIA, M.D.

THE MANAGEMENT OF MENTAL DEFICIENCY IN CHILDREN. By I. Newton Kugelmass, B.S., M.A., M.D., Ph.D., Sc.D. Cloth. Illustrated. Pp. 312. Price $6.75. New York: Grune & Stratton, Inc., 1954.

This book constitutes an excellent classification of mental dis-

orders of childhood. It attempts to bring order into a most complex and diverse subject. The only valid criticism one might have is that the book is mistitled, since only some seventy pages are devoted to the management of mental disorders and the remainder of the book to diagnosis, classification, etc. The book nonetheless is of great value. MICHAEL A. BRESCIA, M.D.

HEARING THERAPY FOR CHILDREN. By Alice Streng, M.A., Waring J. Fitch, M.A., Le Roy D. Hedgecock, Ph.D., James W. Phillips, M.D. and James A. Carrell, Ph.D. Cloth. Pp. 371. Illustrated. Price $6.75. New York: Grune & Stratton, Inc., 1955.

This book is primarily intended for the teacher who is to be specially trained to teach the child with a hearing handicap. The subject is well presented and illustrated. The problem is properly introduced by a review of the anatomy involved and the more common causes of hearing impairment. Early case finding is emphasized as well as the social implications to the child of a hearing loss. The appendix to the book contains a bibliography as well as a listing of the various hearing rehabilitation centers throughout the country. MICHAEL A. BRESCIA, M.D.

PROLONGED NEONATAL JAUNDICE AS GUIDE TO EARLY DIAGNOSIS OF CONGENITAL MYXEDEMA. (Nord. med., 52:1759-1761, Dec. 16, 1954). On the basis of a personal case and cases from the literature Åkerrén calls attention to the not frequent coincidence of neonatal jaundice of physiological type with duration of six weeks or more and congenital myxedema. The association is believed not to be accidental. Acquaintance with the syndrome should facilitate early diagnosis of congenital myxedema and thus allow timely start of treatment. In cases of neonatal jaundice of unusually prolonged duration but of physiological type observation and control are advised until the diagnosis of congenital myxedema can be made or excluded.—*J.A.M.A.*

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